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of the

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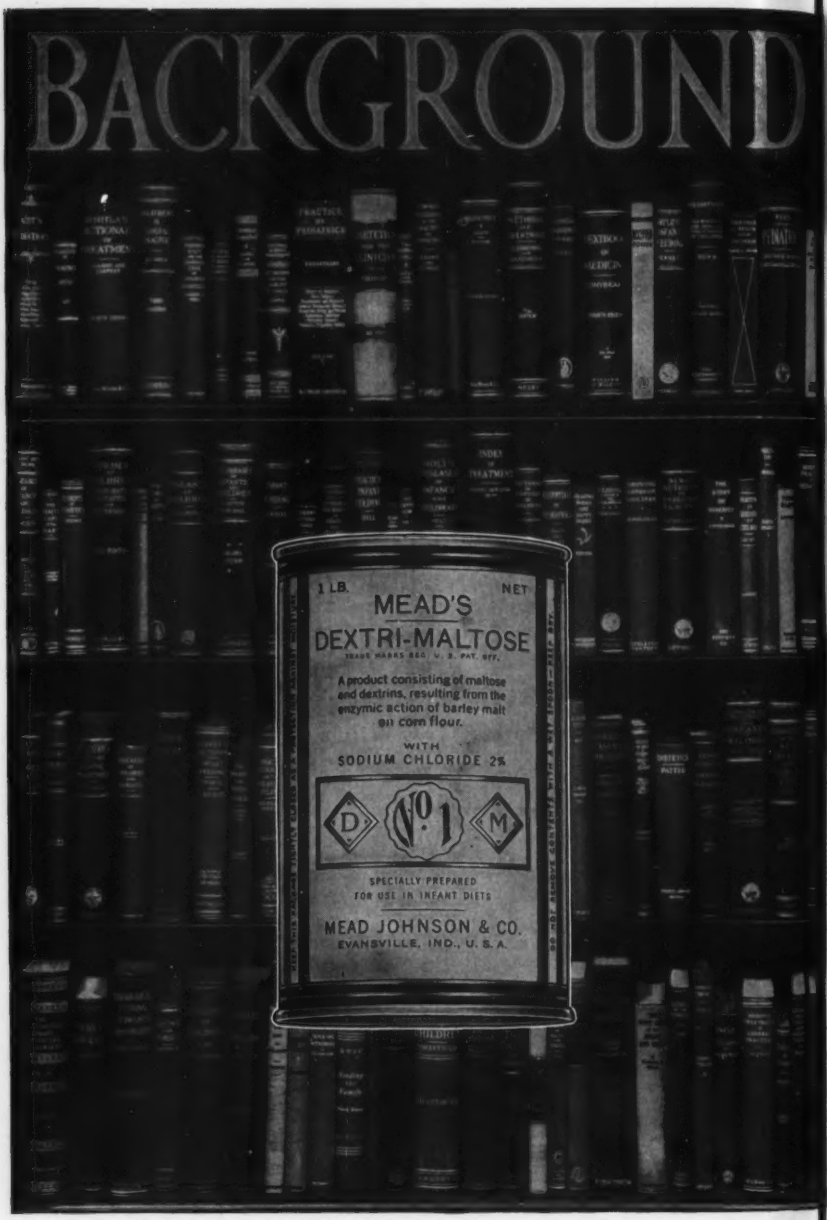

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THE INFLUENCE OF MODERN THERAPEUTIC MEASURES ON THE MORTALITY RATE AT CHILDREN'S HOSPITAL DURING THE PERIOD FROM 1936 THROUGH 1948

E. Clarence Rice, M.D.

The accompanying table of statistics illustrates better than any written words the benefits which the patients at Children's Hospital have received from the recent advances in medicine and surgery. Much of the improvement can be accounted for by the use of chemotherapy, the antibiotics, the increased availability of intravenous therapy and the establishment of our Blood Bank, etc.

DATE	ADMISSIONS	BEDS	DEATHS	AUTOPSIES	%
1936	4617	185	308	157	51.1
1937	4419	185	278	111	62.3
1938	5406	185	213	145	68.0
1939	5504	200	188	135	68.5
1940	6385	200	201	148	73.5
1941	6077	200	250	184	73.6
1942	6237	210	270	172	63.7
1943	6478	210	279	216	77.6
1944	5951	220	266	204	76.6
1945	7094	220	256	208	80.6
1946	8211	225	222	177	79.7
1947	7711	222	195	156	80.0
1948	7755	222	127	109	85.8

The most significant change in our mortality statistics occurred in 1938 when the sulfanilamides first came into general use. From this time on the number of deaths from pneumonia, meningitis, peritonitis, osteomyelitis and similar infections have steadily declined. More recently infantile diarrhea has shown a remarkable lowering of its mortality rate. Some diseases, such as mastoiditis—common 15 years ago, are rarely seen now. Meningitis due to the pneumococcus, streptococcus and Hemophilus influenzae and formerly almost 100 per cent fatal,—can now be treated with the probability of recovery of at least 80 per cent. Rocky Mountain spotted fever, a dread disease, with a mortality rate which previously averaged about 20 per cent, has been treated during the past two years without the loss of a patient.

Much of the recent improvement can be attributed to the development of relatively non-toxic and potent sulfanilamide preparations, such as sulfadiazine, and the increasing array of effective antibiotics, viz., penicillin,

streptomycin, aureomycin and chloromycetin. Blood is now readily available in an emergency for transfusions in most hospitals and the Blood Bank at Children's Hospital, aided by the American Red Cross, has made possible the benefits from this form of treatment. The increasing intravenous use of hydrolyzed protein along with dextrose, sodium chloride and other chemicals has made it possible to maintain nutrition and hydration under conditions which were thought to be hopeless not too long ago.

By studying the causes of death as learned at autopsy the physicians on the staff at Children's Hospital have learned how some obscure conditions can be recognized and various diseases can be treated to better advantage by various medical and surgical means. It has been stated that the quality of medical and surgical work done at a hospital can be evaluated by a perusal of its record of necropsies. Failure to take advantage of the lessons which can be learned at the autopsy table are reflected in an inferior quality of work done. Children's Hospital's record in this respect is outstanding. In 1936 but 51.1 per cent of this material was utilized, whereas in 1948 the percentage had risen to 85.8.

The reduction in the mortality rate has been apparent over the entire country and is reflected in these statistics. In the period from 1936 through 1938 the mortality percentage was 5.5 at this hospital, while in the last three year period this has been reduced to 2.3 per cent. It is believed that of the 23,677 patients who were admitted during this latter period, the lives of at least 700 children were spared from death as compared with the period from 1936 to 1938.

NEUROLOGICAL MANIFESTATIONS OF SHIGELLOSIS

Report of Two Cases

Case Report No. 151

Morris Tandeta, M.D.
Ellsworth Twible, M.D.

In the fall of 1948, at the time the poliomyelitis incidence rate was rather high in this area, many suspect cases were admitted to the hospital for observation and diagnosis. Among these were several patients with neurological signs and prodromal symptoms suggestive of poliomyelitis who after entry began to exhibit diarrhea and prostration to such an extent that the true nature of their illness was soon apparent. The following case reports are typical:

S. C., (48-11648), a 6 year old white male, was admitted on October 8, 1948 with a history of chills and fever of four hours' duration. For one week prior to entry he had had a slight cough, but otherwise was apparently well until the evening of admission, when he suddenly complained of feeling ill, was feverish, and began to have shaking chills lasting several minutes. Nausea and vomiting developed soon afterward.

There was no known exposure to any communicable diseases. The family lived in a new apartment development supplied by the metropolitan water supply and used only pasteurized milk.

Physical examination revealed an acutely ill boy with a temperature of 104° F, pulse 110 per minute, and respirations 23 per minute. Other findings were moderate nuchal resistance to flexion, as well as positive Kernig and Brudzinski signs, and coarse breath sounds audible over the left lower lung field.

Lumbar puncture yielded clear cerebrospinal fluid with no increase in cells or protein. Hemogram revealed hemoglobin 13.5 gms.; erythrocytes 5,000,000; leucocytes 15,400 with 80 per cent neutrophils. Roentgen examination of the chest revealed only considerable perihilar fibrosis.

Approximately ten hours after the onset of his illness the patient began having frequent, loose, bloody, mucoid stools. He became increasingly toxic and at times was irrational. During lucid intervals he complained of abdominal cramps and headache. Stool cultures taken at this time were subsequently reported as having a heavy growth of Shigella sonnei.

Therapy consisted of oral streptomycin, paregoric for the relief of cramps, and intravenous fluids. Twenty-four hours after admission he was entirely rational, his temperature was normal, and he felt well. The diarrhea sub-

sided on the second hospital day. Four successive daily stool cultures were reported as being negative for pathogens. Recovery was uneventful.

L. D., (48-11516), a 6 year old white male, was apparently well until noon of the day of hospital entry, October 4, 1948. He refused his lunch because of nausea and seemed rather listless. In the afternoon, headache and pains in the neck developed. He was taken to a private physician who advised hospitalization because of the possibility of poliomyelitis.

Past history was non-contributory. The family lived in a rural area in Virginia and received its milk supply from a neighbor's cow.

Physical examination revealed a well developed, fairly well nourished white male in no apparent distress. Temperature was 101.8°F., pulse rate 108, blood pressure 110/70, and respirations 25 per minute, The scalp in the right post-auricular area presented a patch of ringworm about 2.5 cm. in diameter with a central encrusted papule surrounded by broken hairs. Slight hyperesthesia of the posterior aspect of the neck was elicited and the post-cervical lymph glands were enlarged. There was marked cheilosis, a slick tongue, herpes simplex of the lips, and slight injection of the nose, throat, and ear drums. Neurological examination at this time was entirely negative, and there were no other significant findings except for a convergent squint.

Lumbar puncture yielded clear cerebrospinal fluid with no increase in cells or protein. Blood for febrile agglutinations, serology, and culture was drawn and reported at a subsequent date as negative. Hemogram revealed hemoglobin 12 gms.; erythrocytes 3,300,000; leucocytes 15,600 with 78 per cent neutrophils. Urinalysis was negative except for 10 mgm. albumin and a few red cells on microscopic examination.

The day following admission the patient began having frequent, loose, foul stools containing mucous shreds. He complained of severe backache and abdominal cramping. His condition rapidly deteriorated as he became quite feverish, dehydrated, toxic, and stuporous.

Re-examination revealed slight nuchal rigidity and a positive Kernig on the right. Stool cultures taken at the onset of diarrhea were subsequently positive for Shigella sonnei.

Therapy consisted of parenteral fluids, including whole blood and plasma, oral streptomycin, and symptomatic measures. In the next 24 hours there was marked general improvement with a return of the temperature to normal. The sensorium gradually cleared. Stool cultures became negative on October 11, the seventh day of illness.

DISCUSSION

Shigellosis has been reported from almost every corner of the globe since Shiga in the latter part of the nineteenth century, in Japan, first isolated the

dysentery organism representative of the genus now bearing his name[1]. It is endemic in this country, but does not occur usually in epidemic form with explosive onset, fulminating course, and high mortality rate, though serious outbreaks now and then make their appearance[5]. Bacillary dysentery is associated with filth, poverty, and crowding. It has often been described as a disease of the poorer classes, and is almost entirely spread by humans, whether in carrier state or actively infected[7].

In infants and young children, shigellosis has perhaps its greatest incidence, especially that due to S. sonnei. Although infection with the Sonne strain is supposed to be milder than that due to others of the group, it can vary from an asymptomatic form or mild diarrhea to the most severe dysentery[6]. Incidence is greatest from about May through October, though sporadic cases may occur at any season of the year.

During outbreaks when acute diarrheal diseases are encountered there is no difficulty in diagnosis. Even in mild cases of diarrhea repeated rectal swabs and culture on multiple selected media such as S.S. agar, desoxycholate citrate agar, and tetrathionate broth aid in bacteriological isolation of any pathogens present. On the other hand, the atypical isolated case with abrupt onset, high fever, prostration, convulsions, toxemia, signs of meningeal irritation, delirium, and coma without preceding diarrhea may offer a confusing diagnostic problem[8]. Involvement of the central nervous system is ruled out by lumbar puncture, though cerebrospinal fluid pressure may be increased because of the cerebral edema. Whether cerebral edema is due to general toxicity or a neurotropic strain of the dysentery organism is not known. Dodd et al.[2] suggest that an underlying nutritional and chemical deficiency, namely hypocalcemia, is at the basis of this symptom-complex, which is elicited by fever and dehydration. That this mechanism is operative in the above cases would be difficult to prove, since their neurological signs were evidenced before the actual onset of diarrhea. Yet, the second patient, L. D., manifested other signs of nutritional deficiency, so that in this instance, at least, it may have had some bearing on the development of his symptomatology.

The onset of diarrhea with resulting dehydration, acidosis, and prostration, suggested the correct diagnosis. After vigorous parenteral therapy was started there was rapid improvement. The gastro-intestinal tract infection was satisfactorily cleared by the administration of oral streptomycin.

SUMMARY

This is a report of two patients admitted to the hospital during the peak of the poliomyelitis season as suspected cases. The true nature of their illness was manifested within twenty four hours. A brief discussion of neurological manifestations in shigellosis is presented.

BIBLIOGRAPHY

1. Bradford, W. L.: Bacillary Dysentery. Mitchell-Nelson Textbook of Pediatrics. pp. 376–380. W. B. Saunders Company, Philadelphia, 1947.
2. Dodd, K., Buddingh, G. J., and Rapaport, S.: The Etiology of Ekiri. Pediatrics, 3, pp. 9–18, January, 1949.
3. Felsen, J.: Recent advances in bacillary dysentery. N. Y. State J. Med., **42:** pp. 789–793, April 15, 1942.
4. Felsen, J. and Osofsky, A. G.: Sonne dysentery. J. A. M. A., **103:** pp. 966–971, September 29, 1934.
5. Felsen, J., Rundlett, E. V., Sullivan, J., and Gorenberg, H.: Atypical Flexner Dysentery—A Preliminary Report of the Jersey City Epidemic. J. A. M. A., **103:** pp. 1055–1058, October 6, 1934.
6. Hardy, A. V. and Watt, J.: Shigellosis. Brennemann's Practice of Pediatrics, Volume II, Chapter 5. W. F. Prior and Company, Hagerstown, Maryland, 1948.
7. Ouyand, G.: Acute bacillary dysentery in infants and children. J. Trop. Med. & Hyg., **44:** pp. 54–59, 1941.
8. Thompson, C. M., White, B. V., Jr., and Schafer, W. L.: Shigellosis studies—etiology and clinical features, U. S. Nav. Med. Bul., **46:** pp. 528–540, April 1946.
9. White, B. V., Jr., Thompson, C. M., Schafer, W. L., and Jacobson, M. A.: Shigellosis studies. U. S. Nav. Med. Bul., **46:** pp. 704–708, May 1946.
10. Thompson, C. M. and White, B. V., Jr.: Shigellosis studies—clinical observations on dysentery caused by Shigella flexneri III, U. S. Nav. Med. Bul., **46:** pp. 901–911, June 1946.

EMPYEMA AS A COMPLICATION OF MEASLES

Case Report No. 152

Joseph M. Lo Presti, M.D. R. R., (48-14638)

R. R., a six year old white male was admitted to Children's Hospital on December 20, 1948. He entered the hospital with the history of having developed a generalized morbilliform rash over the entire body four days prior to admission. Concomitantly, a croup-like cough and a fever of 103°F. was noted. The following day the patient appeared to improve; however, two days prior to admission, he regurgitated his breakfast and developed marked anorexia. The fever and anorexia had persisted up until the day of admission. On the day of entry, shortness of breath was noted and the patient developed diffuse, cramp-like abdominal pain.

The past history was essentially negative except for the presence of intermittent enuresis. The patient had had uncomplicated chicken-pox in February of 1947. Early development had been normal. Vaccination and routine prophylactic inoculations had been carried out in infancy. The family history was non-contributory. No history of exposure to measles was elicited.

The physical examination on admission revealed the temperature to be 100.2°F.; the pulse rate was 108, and the respirations were 50. The patient was a well-developed, well nourished, white male child who was lying quietly in bed and who was obviously acutely ill. The respirations were labored and rapid; breathing was abdominal and intercostal retractions were prominent. A diffuse, confluent, maculopapular rash was present involving the entire body with the exception of the palms and soles. The lips and tongue were dry and crusted. There was moderate injection of the pharynx and tonsils. Examination of the chest revealed dullness to percussion with depression of breath sounds over the left, lower lung posteriorly. Cardiac examination was negative. The abdomen was tense and flat presenting diffuse tenderness throughout all quadrants. The remainder of the physical examination was essentially negative. Admission impressions were rubeola and bronchopneumonia.

For the next two days examinations revealed a dull percussion note and almost complete absence of breath sounds over the lower lobe of the left lung posteriorly. A pleural friction rub in this area was heard by one observer. On admission the hemoglobin was 12 grams; the erythrocytes numbered 4,000,000 and a leukocytosis of 22,000 was present with 70 per cent polymorphonuclear forms. At the time of entry, an X-ray of the chest revealed the heart to be shifted to the right. An opacity present over the

lower two-thirds of the left lung was interpreted as having the appearance of consolidation with a small amount of fluid (Fig. 1). A urinalysis was negative. The patient became progressively more dyspneic and was placed in an oxygen tent. Repeated hemograms revealed a persistent leukocytosis

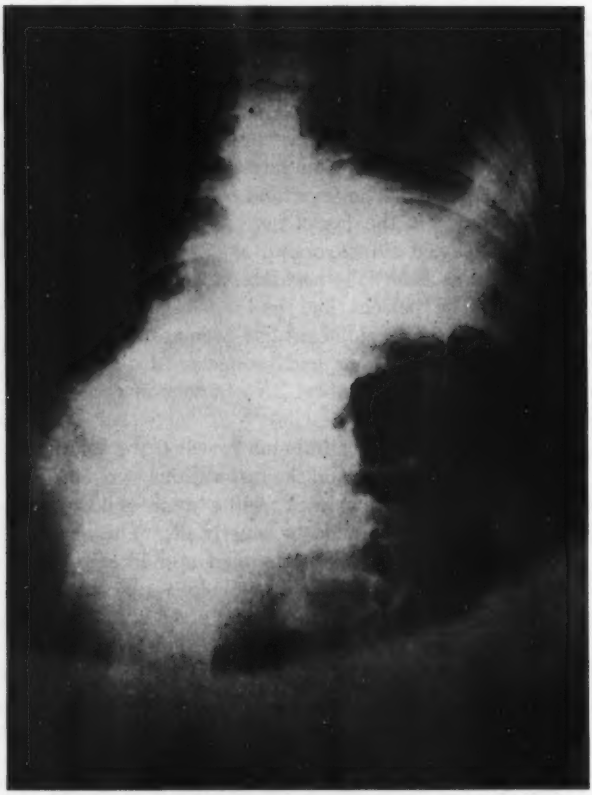

Fig. 1. Admission x-ray. There is an opacity present over the lower two-thirds of the left lung interpreted as having the appearance of consolidation with a small amount of fluid.

varying from 15,000 to 30,000 white blood cells, with a predominance of neutrophiles ranging from 68 to 84 per cent.

Initial therapy consisted of parenteral fluids, mild sedation, and procaine penicillin in oil, 300,000 units daily. On the third hospital day, when an X-ray revealed a massive, left pleural effusion (Fig. 2), a diagnostic thora-

centesis was performed in the left posterior axillary line in the region of the 8th interspace. Three cubic centimeters of turbid, amber fluid was withdrawn from the chest. This fluid was reported as having 54,000 leukocytes per cubic centimeter. Pneumococcus (type IV) and a gram negative,

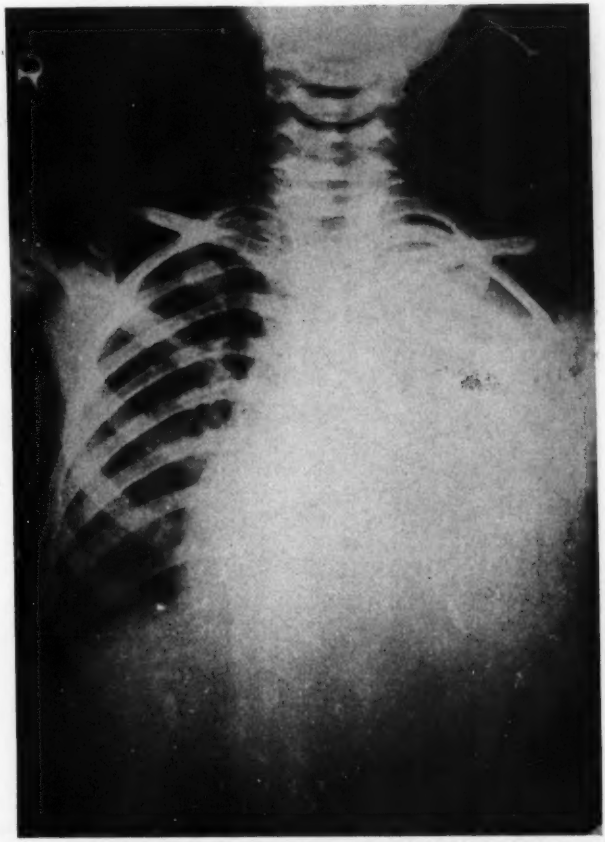

Fig 2. Third hospital day. X-ray reveals a massive left pleural effusion.

pleomorphic bacillus were cultured from the pleural fluid. One-half gram of streptomycin and 500,000 units of penicillin were instilled into the chest. The patient was also placed on parenteral streptomycin, in a dosage of 0.125 grams every three hours. He began to complain of severe chest pain which necessitated sedation with codeine. On the fifth and tenth hospital

days, thoracenteses were performed with the removal of 550 and 600 cubic centimeters respectively of thin purulent fluid. Each of these were followed by the instillation of 100,000 units of penicillin and 0.5 grams of streptomycin into the left pleural cavity. Smears and cultures of the chest fluid obtained from the second and third thoracenteses were reported as negative.

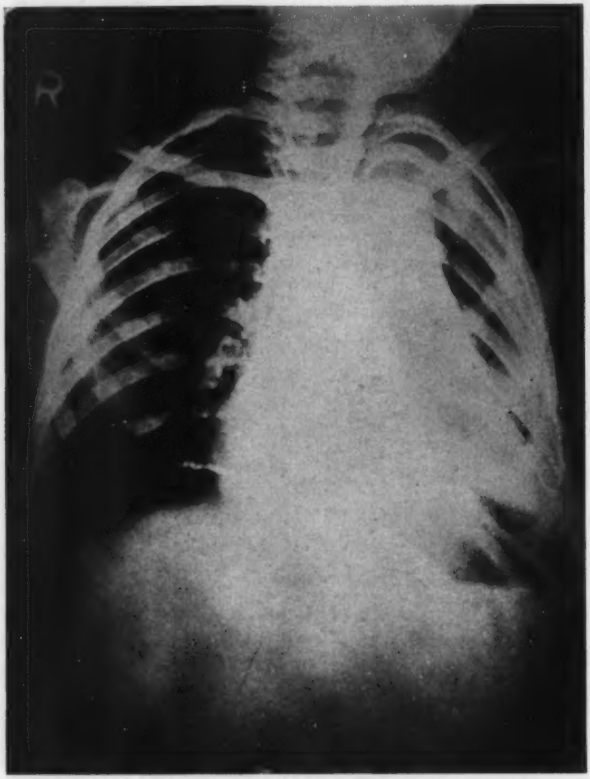

Fig. 3. X-ray following surgical drainage and the incident when the drainage tube was found out of the water. This revealed a left pneumothorax.

On the third of January, fifteen days after admission, the ninth left rib was resected and tube drainage of the empyema instituted. The streptomycin was discontinued, and the parenteral penicillin changed to crysticillin, 300,000 units, three times a day. On the day following operation, the oxygen was discontinued. The patient progressed favorably and his appe-

tite improved slowly. The temperature which had been spiking to 102°F. now came down by lysis, the highest elevation being 100.4°F. However, four days after operation, the tube which drained the empyema was found out of the water. Following this incident, a roentgenogram revealed the presence of a left pneumothorax (Fig. 3). The child began to run an inter-

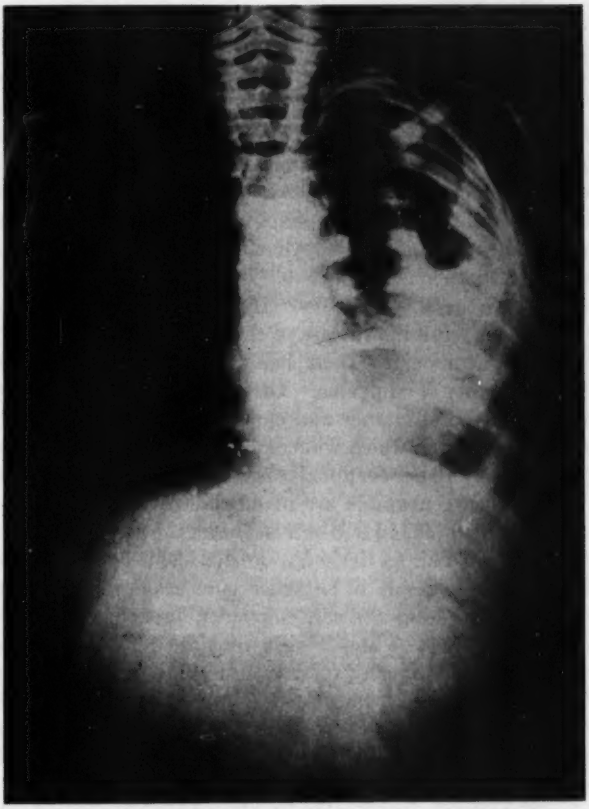

Fig. 4. X-ray on discharge which reveals marked pleural thickening on the left and an area of pneumothorax overlying a fluid level in the middle third of the left lung field.

mittent type of temperature course, with afternoon and evening spikes up to 101°F. Deep breathing, coughing, and blowing up toy balloons were encouraged in an attempt to re-expand the collapsed left lung. By the third of February, the forty-sixth hospital day, the closed drainage had been discontinued, and a small tube was inserted into the chest in place of

the large tube. The patient was permitted to be out of bed. Radiography at that time revealed marked pleural thickening on the left and an area of pneumothorax overlying a fluid level was noted in the middle third of the left lung field (Fig. 4). However, the child's general condition had remained good; he was relatively afebrile, his appetite was excellent, and he appeared to have gained some weight. On the fifth of February, forty-eight days after admission, he was discharged in the care of his private physician.

DISCUSSION

With the recent advances in medical therapeutics, there has been a noticeable and steady decline in the complications and sequelae which, at one time, were so common to certain disease entities. It is to be noted that some of these complications are becoming medical oddities. In the era of medicine which has just passed, empyema was a fairly common complication of infective pulmonary processes, particularly of those due to the pneumococcus and the streptococcus. However, in the modern hospital, one only rarely sees a full-blown case of empyema following pneumococcal or streptococcal pneumonia. There has therefore, been an absolute decrease in the morbidity of empyema. On the other hand, severe measles is still a common disease of infancy and childhood. No specific therapeutic agent has been discovered which will combat this disease. Pneumonia occurs in approximately 10 per cent of all cases and is responsible for nearly all fatalities[6]. If roentgenograms are made routinely, pneumonic infiltrations are found in about one-fourth of the cases in the pre-eruptive stage and in approximately one-half as the eruption subsides[7]. Measles bronchopneumonia appears to result in a higher percentage of other secondary complications such as lung abscess, empyema, bronchiectasis, and unresolved pneumonia[11]. The importance and danger of the pulmonary complications which accompany rubeola, especially in infancy, cannot be stressed too strongly. It becomes readily apparent, therefore, that there has been a relative increase in the incidence of empyema as a complication of measles. Indeed, the last three cases of empyema known to the writer have occurred in conjunction with this disease.

No one can gainsay the tremendous strides which have been made in the field of immunization. Immune globulin and convalescent serum have proven their efficacy in the prophylaxis and modification of measles. Of all of the contacts who receive early immunization, severe symptoms develop in approximately one per cent, and the occurrence of severe complications is practically nonexistent[3, 4, 10]. Some observers present evidence that when convalescent serum is used therapeutically, late in the course of illness, the symptoms will be attentuated and complications prevented[3].

However, convalescent serum is difficult to obtain and must be administered in large amounts. Too, there is no substantial evidence that such a beneficial effect is exerted.

World War II offered an excellent opportunity to study the effects of mass chemoprophylaxis. At first the results were promising[2, 5], so much so, that it has become the policy of some practitioners to routinely prescribe small doses of sulfadiazine in the management of all of their measles patients. Such a policy is practiced in the hope that protection against secondary invaders has thus been attained. The evidence has rapidly accumulated that such mass or individual chemoprophylaxis is not justified[12, 13]. It has been shown that some strains of bacteria are inherently resistant to the action of these drugs. Other strains of susceptible bacteria soon develop "drug-fastness", particularly when small doses are employed. This does not represent a condemnation of chemoprophylaxis, but it is an appeal that chemoprophylaxis be reserved for those outbreaks in which streptococcal sequelae are prevalent. It therefore becomes logical to postulate that the effective control of secondary complications rests in the administration of therapeutic dosages of the bacteriocidal and bacteriostatic drugs which are now at the disposal of the medical profession. If such is the case, and the writer feels that it is, then either full dosages of sulfonamide or penicillin should be used routinely in the management of all cases of measles. The sulfonamide drugs have their limitations. The danger of drug reactions when weighed against the benefit which might be derived from the use of sulfonamides mitigates against this policy. On the other hand, the recent development of penicillin products which maintain therapeutic blood levels for periods as long as seventy-two hours seem to make them the ideal preparations to employ for such a purpose. Further investigation along these lines are necessary before a dogmatic recommendation can be made. The suggestion that these long-acting preparations be employed in the management of every case of measles is offered with the reservation that sufficient evidence of their benefit is still forthcoming. However, penicillin does have a greater safety margin than any of the sulfonamides and is as effective against the common secondary invaders which are encountered during the course of measles.

In the treatment of empyema in its early stages, it is now generally agreed that aspiration is the method of choice. Even before the days of chemotherapy many patients were successfully treated by aspiration alone [8]. The parenteral use of chemotherapy and antibiotics with the instillation of the antibiotics into the pleural cavity following aspiration are probably the most effective methods of therapy. Even with the addition of drug therapy, it occasionally becomes necessary to institute surgical drainage [9]. The indications for surgical drainage are:

1. The development of a bronchopleural fistula as evidenced by coughing up of copious amounts of purulent material. This is a surgical emergency.
2. Toxic symptoms due to the empyema which persist despite adequate medical therapy.
3. The chronic, neglected cases where the involved lung is bound down by fibrous adhesions. In these cases further operative intervention in the form of thoracoplasty is often necessary.

Closed surgical drainage is the method of choice in the cases of diffuse or large loculated empyemas. Open drainage is reserved for small, encapsulated empyemas and in those cases where closed drainage proves inadequate. To insure adequate drainage, a large tube should be inserted into the chest wall. In infants and children this necessitates the removal of a small portion of rib [1]. The post-operative care of a patient with closed drainage for empyema must never be neglected. The house staff and nursing personnel caring for the patient should be given detailed instructions. Particular emphasis on the following points should be made:

1. The free end of the drainage tube must always lie below the level of the water in the collecting reservoir.
2. The drainage tube must be clamped off whenever it becomes necessary to empty the reservoir.
3. The reservoir and tube should never be elevated above the level of the patient's chest.
4. The entire drainage system should be checked every half hour to insure its proper functioning.

The neglect of any of these points can result in disastrous consequences. In the case presented, further operative interference in the form of a thoracotomy may still be necessary before a permanent cure has been effected.

SUMMARY

1. A case of empyema complicating measles is presented.
2. The relative increase in the incidence of empyema as a secondary complication of measles is pointed out.
3. The routine use of any of the long-acting penicillin preparations in the management of all cases of measles is suggested.
4. The precautions to be observed in the post-operative care of a patient who has a closed surgical drainage for empyema are listed.

BIBLIOGRAPHY

1. Burnett, W. E.: Mitchell and Nelson's Textbook of Pediatrics, p. 805 (4th Ed.).
2. Coburn, A. F.: The prevention of bacterial tract infections by sulfadiazine prophylaxis in the United States Navy, J. A. M. A., **126**: 88 (1944).
3. Dungal, N.: Convalescent serum against measles, J. A. M. A., **125**: 20 (1944).

4. Greenberg, Frank, and Rutstein: Gamma globulin and placental globulin. A comparison of their effectiveness in the prevention and modification of measles, J. A. M. A., **126:** 944 (1944).
5. Holbrook, W. P.: The Army Air Forces rheumatic fever control program, J. A. M. A., **126:** 84 (1944).
6. Karelitz, S.: Holt and McIntosh's; Holt's Diseases of Infancy and Childhood, pp. 1134 (11th ed.).
7. Kohn and Koiransky: Successive roentgenograms of the chests of children during measles, Am. J. Dis. Child., **38:** 258 (1929).
——— Further roentgenographic studies of the chests of children during measles, Am. J. Dis. Child., **46:** 40 (1933).
8. McEnery and Brenneman: Aspiration in empyema of children, J. A. M. A., **93:** 362 (1929).
9. Penberthy and Benson: Management and treatment of empyema in children Am. J. Surg., **39:** 267 (1938).
10. Stillerman, Marks and Thalheimer: Prophylaxis of measles with convalescent serum. Principle factors influencing the results, Am. J. Dis. Child., **67:** 1 (1944).
11. Stokes, J., Jr.: Mitchell, and Nelson's Textbook of Pediatrics, p. 446 (4th Ed.).
12. Swyer, R.: Use of sulfonamide in measles, J. Dis. Child., **40:** 63, (1943).
13. Toomey, J. A.: Chemotherapeutics in pediatrics, Kentucky M. J., **43:** 37 (1945).

MEGACOLON

Case Report No. 153

Morris Tandeta, M.D. S. B. 46-5982

S. B., a 20 month old colored male, was admitted to Children's Hospital on April 28, 1947 with complaints of pain following defecation and tenderness in the left lower abdomen. He had been having pain after bowel movements for approximately 10 months and had always been constipated. He was seen in the dispensary at the time of onset of these symptoms and a diagnosis of multiple anal fissures with excoriation of the buttocks was made. Following treatment with 10% silver nitrate the fissures healed, but there was no relief of the pain following bowel movements. Though this pain was not always present, it tended to recur frequently. On a subsequent dispensary visit a "hemorrhoidal tag" or polyp was found. In the month prior to hospital entry the pain was present every day, and there seemed to be tenderness in the left lower abdomen evidenced by crying when the abdomen was touched during bathing or diaper change. At this time the stools were described as yellow-brown, of normal consistency, and as frequent as 2–3 daily. No episodes of vomiting or diarrhea were noted. The patient had been referred to the hospital previously from the welfare clinic because of failure to gain weight, irritability, anemia, and inability to walk or talk.

Past history revealed the patient to be the product of a sixth full term pregnancy with uneventful delivery and neonatal period. He had been breast fed only one month, following which he took an evaporated milk formula, but his appetite was always poor. Orange juice and cod liver oil were given in adequate amounts since he was a month old. The patient's first teeth appeared at 9 months, but at 20 months he was still unable to talk or walk. Family history was non-contributory.

On physical examination the patient was a well developed, poorly nourished, 20 month old colored male with a tremor of the extremities and apparently nervous and apprehensive. There was slight dehydration and loss of subcutaneous fat. The abdomen was moderately distended with some bulging in the flanks. The intestinal pattern was discernible, and there was a slight cylindrical swelling at the left border of the rectus for about 3–4 centimeters below the level of the umbilicus. At the right of the umbilicus and extending to the pubis was another irregularly shaped cylindrical mass which felt superficial, hard, but did not appear tender. Rectal examination revealed a large amount of yellow-brown feces. Neurological examination was not completely successful, though the reflexes were all hyperactive.

Following cleansing enemata the abdominal distention disappeared, as did also the cylindrical masses, except for a small suprapubic mass which was now felt deep in the abdomen, was freely movable, firm, non-tender and was inconstantly palpated by the examiners. Surgical consultation was secured and an exploratory laparotomy was advised to rule out a pelvic tumor.

Laboratory data revealed a mild anemia, for which the patient was given a transfusion of whole blood. Serological test for syphilis was negative. Roentgenological studies were made, including a flat plate of the abdomen, intravenous pyelography, and examination of the long bones. The abdominal film revealed displacement of the large intestine upwards with absence of bowel in the left lower quadrant, but there were no other pathological findings.

On May 2, 1947, the 5th hospital day, exploratory laparotomy revealed a large, dilated, atonic sigmoid colon, approximately 4 times normal size, displacing the bladder to the right. The abdomen was closed in layers and the immediate post-operative condition was satisfactory.

On the 8th post-operative day, as the skin sutures were being removed, the patient eviscerated, and was immediately taken back to the operating room where the wound was closed with through and through wire sutures. Following this he was again transfused with 150 cc. of whole blood. The first spontaneous bowel movement occurred following evisceration.

During the period of convalescence the patient was examined by the Gesell scale and was found mentally retarded.

The wound was quite slow in healing and at first appeared to bulge slightly at the lower end. Reinforcement was possible with adhesive strappings. The wire sutures were finally removed on July 5, 1947. The patient was discharged July 28, 1947.

DISCUSSION

Megacolon or Hirschsprung's disease is a rare affection of the colon, usually the left half, characterized by enlargement of the abdomen with constipation present from early infancy. It is thought that the chief etiologic factor is an imbalance of the autonomic nervous system supplying the colon, although obstructive lesions distally may play a part in some cases. The involved colonic segment, most frequently the sigmoid, is greatly distended with feces and the wall is much thicker than normal because of muscular hypertrophy. X-ray studies and fluoroscopy reveal a dilated colon with loss of normal haustrations and greatly reduced peristalsis.

In more advanced cases the extremities appear wasted and peristaltic patterns become visible through the thinning abdominal wall. Distention may become so great that the diaphragm is displaced upward with embar-

rassment of respiration. Occasionally there is diarrhea, incontinence and overflow of feces. An uncommon complication is acute intestinal obstruction. Inspissated fecal matter may cause ulceration of the mucosa or even perforation of the colon with peritonitis. Terminally the breath has a fetid odor and there is anorexia, lassitude and cachexia.

Although there is a tendency to remissions and exacerbations in megacolon, nutrition, growth, and development are bound to be affected to some extent. Therapy in recent years has done much to prolong life and ameliorate the symptoms of this condition.

In mild forms of megacolon there should be a trial medical management consisting of a 1. cleansing enemate, 2. daily laxatives such as mineral oil, 3. highly nutritious and low residue diet, 4. bowel training, 5. drugs, usually parasympathetic stimulants as acetyl-beta-choline and prostigmine. If the latter are not effective, sympathomimetic drugs such as ergotamine may prove of some value. Mechanical relief by dilatation of a tight anal sphincter has been of benefit when this has caused obstruction to passage of feces.

Surgery is indicated in severe cases not responding to conservative measures. There are three accepted surgical procedures which have proven of value in properly selected cases. Cecostomy has been accomplished successfully when acute intestinal obstruction complicates the primary disease. Colectomy or resection of a loop of dilated large bowel can be done if the involvement is of limited extent. Sympathectomy offers an attack at the basis of the condition. Spinal anesthesia, sometimes used as a test before sympathectomy is attempted, has also been reported as initiating spontaneous bowel evacuation.

Since evaluation of therapeutic results is so difficult, any measure of proven benefit is a boon in this condition for which there is as yet no permanent cure.

CLINICO-PATHOLOGICAL CONFERENCE

Directed by: E. Clarence Rice, M.D.
Assisted by: Edwin Vaden, M.D.
D. Joseph Judge, M.D.
By Invitation: William F. Burdick, M.D.

Edwin Vaden, M.D. P. P. 38-9657

A four weeks old white male entered Children's Hospital with a history of persistent vomiting of three days' duration.

The infant was delivered in a local hospital at term, following an uneventful pregnancy. The birth weight was 7 pounds 6 ounces and he weighed 7 pounds 10 ounces at the time he was discharged approximately ten days after delivery. He had been on an evaporated milk formula which he seemed to take well, although it was said that there was usually a small amount of "spitting" after each feeding.

The infant had been in apparent good health until a week previously when he became somewhat constipated. Three days prior to admission he began vomiting after each feeding. The vomiting usually occurred about two hours after each feeding. It was not projectile in type. Instead, it was described as a sort of "welling up at the mouth." The patient had two bowel movements on the day preceding admission. The morning stool was rather large, liquid and brown, the second one was semi-solid and brown.

Physical examination revealed a fairly well developed one month old baby weighing 7 pounds 8 ounces. The rectal temperature was 99.5°F. The skin was dry and inelastic. The only other positive physical findings were those of the abdomen. A moderate amount of distention was present. Two examiners stated that a small tumor mass was present in the right upper quadrant and that "peristaltic waves are present but they are not altogether typical of pyloric stenosis."

The admission diagnosis, congenital hypertrophic pyloric stenosis, was confirmed at laparotomy, and a Ramstedt procedure was done 24 hours after admission.

The patient's post-operative course was fairly smooth. He was afebrile and the wound healed well. For the first day after surgery he was offered small amounts of boiled water and on the second day he was given evaporated milk formula with water in the proportion of one part formula to three parts water. The proportion of water was decreased until he was receiving straight formula on the fifth post-operative day. However, he persisted in vomiting small amounts after each feeding. Yet his weight, which dropped

to 7 pounds 4 ounces after surgery, rose to 7 pounds 6 ounces on the seventh day. During this period he had one natural bowel movement and daily enemata. He was then placed on thickened feedings which he retained well and he was discharged to his home in satisfactory condition.

For the first two weeks after discharge he retained his feedings and made a small weight gain. Then he began to vomit again and for one week he vomited regularly after each feeding. He was readmitted at two months of age weighing 7 pounds 6 ounces. The temperature was 101.8°F. A urinalysis was normal. The leucocyte count was 15,500 with 49 per cent neutrophiles (33 per cent segmented, 13 per cent bands and 1 per cent young forms), 50 per cent lymphocytes and 1 per cent monocytes.

The vomiting usually occurred during or immediately after each feeding although sometimes he went for several hours before vomiting. The stools were small and hard. A gastro-intestinal x-ray series was done. The scout film showed the stomach to be distended with air. After the swallowing of bismuth about 40 per cent was retained in the stomach at two and three hours, and that which had passed into the small bowel was observed to have gone through to the cecum without difficulty.

A second laparotomy was performed shortly after admission.

DISCUSSION

William F. Burdick, M.D.: The history as given would indicate that this patient had hypertrophic pyloric stenosis. In fact, the record states that this diagnosis was *confirmed* at laparotomy and that the routine Ramstedt procedure was done. I suppose it would be too much for me to expect that this was the correct diagnosis, or indeed the complete diagnosis for he did not do well, became obstructed again, or partially so at least, and had to be operated upon the second time.

A discussion of vomiting and its causes would be a reasonable way to approach the diagnosis, inasmuch as vomiting was his chief complaint.

CAUSES OF VOMITING IN THE NEWBORN INFANT

Faulty feeding of infants. Vomiting is a common symptom during the newborn period and usually simply means a spitting up or regurgitation from over-feeding. Vomiting during the first few hours after birth is frequently due to irritation from swallowed amniotic fluid. When it is persistent, we must consider the possibility of some more serious disturbance. It often results from faulty feeding techniques or improper diets. Too much fat in the diet of very young babies delays gastric emptying, causes distention, abdominal discomfort and vomiting. The vomiting of overfeeding usually occurs shortly after feeding, and has principally a nuisance value. Other causes are too tight clothing about the abdomen, head in a

dependent position or the infant being placed on his back or left side immediately after feeding, instead of on his abdomen or on his right side. In general, the greater the time which has elapsed between the feeding and vomiting, the more serious is apt to be the underlying cause.

Intracranial injury or hemorrhage which results from birth trauma with an increase in intracranial pressure may cause vomiting in the newborn. There may be a frank hemorrhage, or simply cerebral edema. The signs and symptoms are similar in each case, but the Moro reflex may be of value in making the differentiation. The Moro reflex is absent early, but present after a few days when the edema has subsided. If there is cerebral hemorrhage the Moro reflex may be elicited immediately after birth, but is absent later if there is cerebral damage. The complete absence of the Moro reflex in a newborn infant is highly suggestive of cerebral damage. The vomiting of intracranial injury is likely to be in small amounts and it appears at irregular intervals, unrelated to the ingestion of food.

Pylorospasm. Babies with pylorospasm are apt to have generalized muscle hypertonicity of the trunk and extremities. The vomiting occurs in spells, that is the child will vomit for two or three days and then be spontaneously relieved for several days before vomiting reappears. No abdominal tumor is palpable. Pylorospasm can be relieved by using atropine compounds. Pylorospasm must be differentiated from pyloric stenosis.

Acute infectious diseases. Vomiting is a common initial symptom of most of the acute infectious diseases. This patient upon his return to the hospital the second time had fever and leucocytosis, so perhaps had some infection but no localizing or specific symptoms are given in the history. The common cold, as well as pneumonia, scarlet fever, etc., may be ushered in with vomiting.

Acute gastro-intestinal diseases. Vomiting may be a prominent symptom in all diarrheal disturbances such as dysentery and salmonella infections as well as parenteral diarrhea and those of unknown etiology.

Appendicitis. Vomiting is an early symptom in acute appendicitis and becomes manifest again in the case of rupture. The symptom is more marked if generalized peritonitis occurs than if the appendiceal abscess localizes.

Acute peritonitis. In contrast to chronic peritonitis, vomiting is a constant symptom and is perhaps due to paralytic ileus accompanying the peritonitis.

Intussusception. The vomiting which accompanies intussusception as in appendicitis is preceded by intermittent colicky pain. A horse-shoe or sausage shaped mass may be palpated in the abdomen, and the gloved finger may reveal blood and mucous in the rectum. The vomiting is persistent, and if the intussusception is unrelieved the vomitus will contain intestinal contents, the nature depending on the level of the obstruction.

Peptic ulcer. Peptic ulcer may infrequently be a cause of vomiting in infants and children. If the ulcer occurs in the pyloric ring the vomiting may be projectile in type. Blood is usually encountered either in the vomitus or in the stools in such cases. There have been a few cases of peptic ulcer either of the duodenum or stomach discovered at autopsy in this hospital. The vomiting which is present in peptic ulcer usually occurs an hour or two after ingestion of food and it is accompanied by epigastric pain.

Organic nervous disease. Vomiting is an early symptom in all of the meningitides. Space occupying intracranial lesions such as neoplasma, abscesses and hemorrhage frequently cause vomiting. It may be projectile in type, unassociated with nausea and occurs with change in position. It often occurs shortly after the child arises in the morning, which causes an increase in intracranial pressure.

Dysfunction of the adrenal gland. Hypofunction of the adrenal cortex in infants may produce a syndrome almost indistinguishable from intestinal obstruction. Vomiting is one of the characteristic symptoms, and if the true diagnosis is unrecognized, the patient may be subjected to surgery. These cases usually show some brownish skin pigmentation and perhaps enlargement of the external genitalia.

Reflex vomiting. Some children have a particularly sensitive gag reflex. Such conditions as a copious post-nasal drip, enlarged uvula, persistent or spasmodic cough (pertussis), or gagging on food may cause vomiting.

Toxic vomiting. This term is applied to the vomiting which accompanies uremia, diabetic acidosis and certain poisons such as apomorphine, tartar emetic and digitalis which stimulate the respiratory center.

Other causes of vomiting. These include ether anesthetics, operative manipulations of the abdominal organs, and swallowing of caustic poisons.

Congenital hypertrophic pyloric stenosis. The vomiting is projectile in character, usually beginning in the third week of life in a male white child. The pyloric tumor may be palpated, if conditions are favorable, just outside the right rectus muscle between the level of the umbilicus and the costal arch. The patient loses weight and is constipated. If the patient has been recently fed, pyloric waves may be seen rising in the left side of the abdomen and disappearing on the right side at about where the tumor is felt.

The fact that the waves in this case were faint and not typical of pyloric stenosis leads me to believe that this case did not have a true pyloric stenosis. I have never seen a case, proven at operation, where the peristaltic waves were not good big ones and unquestionable. The vomiting in this case was not projectile and I have never seen a proven case at operation that did not previously have forceful unquestionable projectile vomiting. Yet the record says that the diagnosis was confirmed at laparotomy. The

small tumor mass which was felt in this case could have been distended duodenum above the obstructive lesion.

Other forms of congenital obstructive lesions. These lesions may be partial or complete and they may be single or multiple. In complete obstruction the vomiting occurs shortly after birth and no fecal matter is passed. It is obvious in this case that the obstruction was not complete for the signs of partial obstruction appeared after several weeks of life and fecal matter had been passed. I am unable to say whether the lesion was single or multiple. It must have been multiple if the record is true that pyloric stenosis was "confirmed at laparotomy," unless this is a case of recurring pyloric stenosis as was reported recently in the Clinical Proceedings. I doubt if this was such a case.

Obstructive lesions which occur below the ampulla of Vater will produce bile in the vomitus. If the obstruction is lower in the small bowel or colon, the vomitus will contain fecal matter. The distention is in the epigastrium if obstruction exists in the duodenum and there may be gastric peristaltic waves. Lower obstructions are apt to cause distention in the flanks and marked peristaltic activity is present above the lesion.

This case had epigastric distention and some peristaltic waves. There was no bile in the vomitus, so the obstructing lesion must have been in the duodenum above the ampulla of Vater.

The ampulla of Vater is located about 10 centimeters (4 inches) below the pylorus in the middle of the second portion of the duodenum. It is in this portion of the duodenum that I believe the lesion in this case was located. The lesion could be (1) constricting band from without, such as that induced by incomplete rotation of the intestinal tract, or (2) an intrinsic destruction of the lumen by congenital stenosis or constriction. In the former the cecum commonly lies just below the distal half of the stomach and bands of reflected peritoneum run from it to the right posterolateral part of the abdominal wall. These bands or folds therefore lie directly across the descending portion of the duodenum and partly obstruct this viscus by external pressure. My third choice would be that of a recurrent pyloric stenosis.

Doctor Burdick's diagnoses:

1. Incomplete obstruction of duodenum proximal to the ampulla of Vater.
   a. External band.
   b. Congenital stenosis.
2. Recurrent pyloric stenosis.

Doctor Tandeta, Resident in Pediatrics.

I believe we are agreed that the history, physical findings, and subsequent course in the hospital are consistent with the diagnosis of pyloric stenosis

at the time of the first admission. On the second admission, however, I am suspicious of some condition other than an obstruction of the gastro-intestinal tract. From the fever and leucocytosis we may expect some infectious process is taking place which might be responsible for the vomiting. Despite the single negative urinalysis, we cannot rule out genito-urinary disease, whether it be on the basis of a renal anomaly with secondary infection or a primary infection such as pyelitis and I propose some such occult genito-urinary abnormality as the basis for the subsequent illness.

Doctor Cashion, Resident in Pediatrics.

I have seen a recurrent intestinal obstruction with a similar picture caused by a diverticulum which would fill up and then turn over on the intestine so as to produce an obstruction by extrinsic pressure.

PATHOLOGICAL DISCUSSION

E. Clarence Rice, M.D.: At the second operation the surgeon found the pylorus adherent to the surrounding tissues which included the liver. The musculature at the pylorus was about two-thirds the thickness as found at the first operation. The original incision was opened and the muscle split at a different point. The patient seemed to improve following this operation and took his feedings well but two days later vomiting recurred and the temperature rose to 108°F. Death occurred two days after operation.

At autopsy the body was that of a poorly nourished white male. There was considerable venous stasis over the dependent portions of the body.

The pertinent findings were as follows:

The right auricle was dilated and the musculature was flabby. No fluid was present in the pleural or peritoneal cavities. The omentum was adherent to the pyloric portion of the stomach.

The gastro-intestinal tract was distended with gas. The mucosa of the stomach was edematous and along the greater curvature were many sharply demarcated ulcers. Two longitudinal healed operative scars were noted at the pylorus. The incisions did not involve the mucosa. The pyloric muscle was 0.5 centimeters in thickness. There is no evidence that the lumen of the pylorus was obstructed. The mucosa of the small gut was also edematous and in the jejunum a number of scattered areas of hemorrhage, which measure up to 2 centimeters in diameter, were present. There was evidence of old and recent hemorrhage.

Both kidneys were edematous and congested. The right kidney showed a markedly dilated pelvis with a capacity of 20–25 cubic centimeters. The ureter was kinked at the uretero-pelvic junction obstructing the flow of urine. The bladder was thick walled and the mucosa edematous.

PATHOLOGIC DIAGNOSIS

Post-operative pyloroplasty
Ulcerative gastritis
Hemorrhagic enteritis, subacute
Hydronephrosis, left, due to kink in ureter
Bronchial, mediastinal, and mesenteric lymphadenopathy
Congestion of liver, spleen, and kidneys

It would appear that the two operations were sufficient to relieve any obstruction to the passage of stomach contents through the pylorus. It is quite likely that the ulceration of the stomach mucosa served to perpetuate the vomiting. The evidence of hemorrhage in the intestine may have been due in part to a vitamin K deficiency with resulting bleeding tendency.

We have seen a few patients die of peritonitis following pyloplasty following perforation of the duodenum. However, when this accident occurs it is usually recognized by the surgeon and appropriate measures are taken to correct the surgical accident. Occasionally edema of the pylorus following operation will be so great as to prevent the passage of stomach contents.

It seems unlikely that the urinary tract pathology would have caused the recurrent vomiting in this case.

REFERENCES

1. Mitchell and Nelson: Textbook of Pediatrics, W. B. Saunders and Co., 4th Edition, 1947.
2. Ladd and Gross: Abdominal Surgery of Infancy and Childhood, W. B. Saunders and Co., 1947.
3. Chenoweth: Dysfunction of the Adrenal Gland in Infancy, J. South. Med. Ass. **41:** 307 April, 1948.
4. Burdick, W. F.: Peptic Ulcer in Children. J. of Ped., **17:** 654–658, Nov. 1940.
5. Frazier, D. A. and Warfield, J. O.: Recurrent Pyloric Stenosis, Clin. Proc. Child. Hosp., Washington, D. C. (17).